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 NEWS  4  Apr 09  ZDB will be removed from STN
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IFIUDB
 NEWS  6  Apr 22  Records from IP.com available in CAPLUS, HCAPLUS, and
ZCAPLUS
 NEWS  7  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS  8  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS  9  Jun 03  New e-mail delivery for search results now available
 NEWS 10  Jun 10  MEDLINE Reload
 NEWS 11  Jun 10  PCTFULL has been reloaded
 NEWS 12  Jul 02  FOREGE no longer contains STANDARDS file segment
 NEWS 13  Jul 22  USAN to be reloaded July 28, 2002;
                  saved answer sets no longer valid
 NEWS 14  Jul 29  Enhanced polymer searching in REGISTRY
 NEWS 15  Jul 30  NETFIRST to be removed from STN
 NEWS 16  Aug 08  CANCERLIT reload
 NEWS 17  Aug 08  PHARMAMarketLetter(PHARMAML) - new on STN
 NEWS 18  Aug 08  NTIS has been reloaded and enhanced
 NEWS 19  Aug 19  Aquatic Toxicity Information Retrieval (AQUIRE)
                  now available on STN
 NEWS 20  Aug 19  IFIPAT, IFICDB, and IFIUDB have been reloaded
 NEWS 21  Aug 19  The MEDLINE file segment of TOXCENTER has been reloaded
 NEWS 22  Aug 26  Sequence searching in REGISTRY enhanced
 NEWS 23  Sep 03  JAPIO has been reloaded and enhanced
 NEWS 24  Sep 16  Experimental properties added to the REGISTRY file
 NEWS 25  Sep 16  Indexing added to some pre-1967 records in CA/CAPLUS
 NEWS 26  Sep 16  CA Section Thesaurus available in CAPLUS and CA
 NEWS 27  Oct 01  CASREACT Enriched with Reactions from 1907 to 1985

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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=> s gonzalez j/au

L2 2354 GONZALEZ J/AU

=> s l1 and l2

L3 0 L1 AND L2

=> s transmembrane (p) potential (p) ompound (p) fluores? (p) dye

L4 0 TRANSMEMBRANE (P) POTENTIAL (P) OMPOUND (P) FLUORES? (P) DYE

=> s transmembrane (p) potential (p) compound (p) fluores? (p) dye

L5 18 TRANSMEMBRANE (P) POTENTIAL (P) COMPOUND (P) FLUORES? (P) DYE

=> s transmembrane (p) potential (p) compound (p) FRET

L6 1 TRANSMEMBRANE (P) POTENTIAL (P) COMPOUND (P) FRET

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L5 ANSWER 1 OF 18 MEDLINE
ACCESSION NUMBER: 2002199315 MEDLINE
DOCUMENT NUMBER: 21929619 PubMed ID: 11933013
TITLE: Mitochondrial and nonmitochondrial reduction of MTT: interaction of MTT with TMRE, JC-1, and NAO mitochondrial fluorescent probes.
AUTHOR: Bernas Tytus; Dobrucki Jurek
CORPORATE SOURCE: Laboratory of Confocal Microscopy and Image Analysis,

Department of Biophysics, Institute of Molecular Biology and Biotechnology, Jagiellonian University, Krakow, Poland.
SOURCE: CYTOMETRY, (2002 Apr 1) 47 (4) 236-42.
Journal code: 8102328. ISSN: 0196-4763.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020405
Last Updated on STN: 20020801
Entered Medline: 20020731

AB . . . MTT, we imaged the formation of MTT-formazan deposits using backscattered light confocal microscopy. Mitochondria were visualized in viable cells using **fluorescent dyes** that bind in a manner dependent (JC-1 and TMRE) or independent (NAO) of mitochondrial electric **potential**. RESULTS: Only 25-45% of MTT-formazan was associated with mitochondria after 25 min of incubation. No more than 25% of the mitochondrial area on images was occupied by MTT-formazan. Mitochondrial **fluorescence** of TMRE, NAO, and the monomeric form of JC-1 decreased rapidly in cells incubated with MTT. However, the intensity of **fluorescence** of JC-1 aggregates dropped by less than 30% at the onset of incubation and remained constant as reduction of MTT. . . well as TMRE and NAO, accumulating in mitochondria may be displaced by MTT. Thus, the presence of positively charged organic **compounds** (like MTT) may distort measurements of mitochondrial **transmembrane** electric **potential**, which are based on accumulation of **fluorescent dyes**.

L5 ANSWER 2 OF 18 MEDLINE
ACCESSION NUMBER: 2001668756 MEDLINE
DOCUMENT NUMBER: 21571345 PubMed ID: 11714485
TITLE: Mitochondrial injury by disulfiram: two different mechanisms of the mitochondrial permeability transition.
AUTHOR: Balakirev M Y; Zimmer G
CORPORATE SOURCE: Institut de Biologie Structurale, 41 rue Jules Horowitz, 38027 Grenoble, France.. maxbala@ibs.fr
SOURCE: CHEMICO-BIOLOGICAL INTERACTIONS, (2001 Dec 21) 138 (3) 299-311.
Journal code: 0227276. ISSN: 0009-2797.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011121
Last Updated on STN: 20020125
Entered Medline: 20020103

AB Disulfiram (Ds), a clinically employed alcohol deterrent of the thiuram disulfide (TD) class of **compounds**, is known to cause hepatitis and neuropathies. Although this drug has been shown to inhibit different thiol-containing enzymes, the actual. . . GSH-dependent manner. At the concentration above characteristic threshold, TDs induced irreversible oxidation of NAD(P)H and glutathione (GSH) pools, collapse of **transmembrane potential**, and inhibition of oxidative phosphorylation. The presence of Ca(2+) and exhaustion of mitochondrial glutathione (GSH+GSSG) decreased the threshold concentration of TDs. Swelling of the mitochondria and leakage of non-transported **fluorescent dye** BCECF from the matrix indicated that TDs induced the mitochondrial permeability transition (MPT). Mitochondrial permeabilization by TDs involves two, apparently. . .

L5 ANSWER 3 OF 18 MEDLINE
ACCESSION NUMBER: 1998430701 MEDLINE

DOCUMENT NUMBER: 98430701 PubMed ID: 9759901
TITLE: Poly(ADP-ribose) synthetase activation mediates mitochondrial injury during oxidant-induced cell death.
AUTHOR: Virag L; Salzman A L; Szabo C
CORPORATE SOURCE: Division of Critical Care, Children's Hospital Medical Center, Cincinnati, OH 45229, USA.
CONTRACT NUMBER: R01HL59266 (NHLBI)
R29GM54773 (NIGMS)
SOURCE: JOURNAL OF IMMUNOLOGY, (1998 Oct 1) 161 (7) 3753-9.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981029
Last Updated on STN: 19981029
Entered Medline: 19981022

AB . . . investigated whether PARS activation contributes to the mitochondrial alterations in cells exposed to oxidants. Authentic peroxynitrite (20 microM), the peroxynitrite-generating **compound** 3-morpholinosidnonimine, the combination of pyrogallol and S-nitroso-N-acetyl-D,L-penicillamine, as well as hydrogen peroxide induced a time- and dose-dependent decrease in mitochondrial **transmembrane potential** (delta psi(m)) in thymocytes, as determined by flow cytometry using the mitochondrial **potential** sensitive **dyes** DiOC6(3) and JC-1. A time- and dose-dependent increase in secondary reactive oxygen intermediate production and loss of cardiolipin, an indicator of mitochondrial membrane damage, were also observed, as measured by flow cytometry using the **fluorescent dyes** dihydroethidine and nonyl-acridine orange, respectively. Inhibition of PARS by 3-aminobenzamide or 5-iodo-6-amino-1,2-benzopyrone attenuated peroxynitrite-induced delta psi(m) reduction, secondary reactive oxygen. . .

L5 ANSWER 4 OF 18 MEDLINE
ACCESSION NUMBER: 1998141227 MEDLINE
DOCUMENT NUMBER: 98141227 PubMed ID: 9482123
TITLE: Mercuric compounds inhibit human monocyte function by inducing apoptosis: evidence for formation of reactive oxygen species, development of mitochondrial membrane permeability transition and loss of reductive reserve.
AUTHOR: InSug O; Datar S; Koch C J; Shapiro I M; Shenker B J
CORPORATE SOURCE: Department of Biochemistry, University of Pennsylvania, School of Dental Medicine, Philadelphia 19104-6002, USA.
CONTRACT NUMBER: DE10873 (NIDCR)
SOURCE: TOXICOLOGY, (1997 Dec 31) 124 (3) 211-24.
Journal code: 0361055. ISSN: 0300-483X.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980312
Last Updated on STN: 20000303
Entered Medline: 19980305

AB The focus of this investigation was to examine the effects of low concentrations of organic mercuric **compounds** on human monocyte function and to relate these effects to apoptosis. Following exposure of monocytes to 0-5 microM MeHgCl, phagocytic. . . monocyte death was due to apoptosis, a number of flow cytometric studies were performed. Mercury-treated cells exhibited increased Hoechst 33258 **fluorescence**, while maintaining their ability to exclude the vital **dye** 7-aminoactinomycin D. Furthermore, monocytes exhibited changes

in light scatter patterns that were consistent with apoptosis; these included decreased forward light. . . mercury. Mercury-treated cells also exhibited changes in lipid organization within the plasma membrane as evidenced by increased uptake of the **fluorescent** probe, merocyanine 540, and by elevated annexin V binding to phosphatidylserine. Using the **fluorescent** probes DiOC6(3) and rhodamine 123 we noted that within 1 h of exposure to mercury, monocytes exhibited a decrease in mitochondrial **transmembrane potential** (psi m). Since a decreased psi m is associated with altered mitochondrial function, the hypothesis that mercury potentiated reactive oxygen. . . was tested. We noted that treated cells generated ROS, as evidenced by oxidation of hydroethidine and the generation of the **fluorescent** product, ethidium. Finally, since ROS would also lower monocyte reductive reserve, we also measured GSH levels in mercury-treated cells. Chemical. . .

L5 ANSWER 5 OF 18 MEDLINE
ACCESSION NUMBER: 84009618 MEDLINE
DOCUMENT NUMBER: 84009618 PubMed ID: 6619770
TITLE: Correlation of increased intraacrosomal pH with the hamster sperm acrosome reaction.
AUTHOR: Working P K; Meizel S
CONTRACT NUMBER: HD-06698 (NICHD)
SOURCE: JOURNAL OF EXPERIMENTAL ZOOLOGY, (1983 Jul) 227 (1) 97-107.
Journal code: 0375365. ISSN: 0022-104X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198311
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831123

AB . . . mM) at 4 hr did not stimulate AR over control levels, suggesting that the stimulation of AR by the other **compounds** was not directly due to depletion of acrosomal adenosine triphosphate (ATP) or alteration of the acrosomal **transmembrane potential**. The AR also was not stimulated by either DCCD or FCCP added prior to 3 hr of incubation of sperm, whereas both **compounds** were increasingly effective at stimulating AR with increasing length of preincubation of sperm before the addition of the test **compounds**. The intraacrosomal pH of sperm incubated in low [K+] (0.6-0.9 mM) for 3.5 hr rose by at least one pH unit (as measured with the **fluorescent dye** 9-aminoacridine) within 15-30 min after raising extracellular [K+] to 4.2-4.5 mM. The pH rise occurred even in the presence of. . .

L5 ANSWER 6 OF 18 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:267732 BIOSIS
DOCUMENT NUMBER: PREV200200267732
TITLE: Mitochondrial and nonmitochondrial reduction of MTT: Interaction of MTT with TMRE, JC-1, and NAO mitochondrial fluorescent probes.
AUTHOR(S): Bernas, Tytus; Dobrucki, Jurek (1)
CORPORATE SOURCE: (1) Laboratory of Confocal Microscopy and Image Analysis, Department of Biophysics, Institute of Molecular Biology and Biotechnology, Jagiellonian University, ul. Gronostajowa 7, 30-387, Krakow: dobrucki@mol.uj.edu.pl Poland
SOURCE: Cytometry, (April 1, 2002) Vol. 47, No. 4, pp. 236-242. http://www.interscience.wiley.com/jpages/0196-4763/. print.
ISSN: 0196-4763.
DOCUMENT TYPE: Article

LANGUAGE: English
AB. . . MTT, w[redacted]maged the formation of MTT-for[redacted]an deposits using backscattered light confocal microscopy. Mitochondria were visualized in viable cells using **fluorescent dyes** that bind in a manner dependent (JC-1 and TMRE) or independent (NAO) of mitochondrial electric **potential**. Results: Only 25-45% of MTT-formazan was associated with mitochondria after 25 min of incubation. No more than 25% of the mitochondrial area on images was occupied by MTT-formazan. Mitochondrial **fluorescence** of TMRE, NAO, and the monomeric form of JC-1 decreased rapidly in cells incubated with MTT. However, the intensity of **fluorescence** of JC-1 aggregates dropped by less than 30% at the onset of incubation and remained constant as reduction of MTT. . . well as TMRE and NAO, accumulating in mitochondria may be displaced by MTT. Thus, the presence of positively charged organic **compounds** (like MTT) may distort measurements of mitochondrial **transmembrane** electric **potential**, which are based on accumulation of **fluorescent dyes**.

L5 ANSWER 7 OF 18 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:74733 BIOSIS
DOCUMENT NUMBER: PREV200200074733
TITLE: Mitochondrial injury by disulfiram: Two different mechanisms of the mitochondrial permeability transition.
AUTHOR(S): Balakirev, Maxim Yu (1); Zimmer, Guido
CORPORATE SOURCE: (1) Institut de Biologie Structurale, 41 rue Jules Horowitz, 38027, Grenoble: maxbala@ibs.fr France
SOURCE: Chemico-Biological Interactions, (December 21, 2001) Vol. 138, No. 3, pp. 299-311. print.
ISSN: 0009-2797.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Disulfiram (Ds), a clinically employed alcohol deterrent of the thiuram disulfide (TD) class of **compounds**, is known to cause hepatitis and neuropathies. Although this drug has been shown to inhibit different thiol-containing enzymes, the actual. . . GSH-dependent manner. At the concentration above characteristic threshold, TDs induced irreversible oxidation of NAD(P)H and glutathione (GSH) pools, collapse of **transmembrane potential**, and inhibition of oxidative phosphorylation. The presence of Ca2+ and exhaustion of mitochondrial glutathione (GSH + GSSG) decreased the threshold concentration of TDs. Swelling of the mitochondria and leakage of non-transported **fluorescent dye** BCECF from the matrix indicated that TDs induced the mitochondrial permeability transition (MPT). Mitochondrial permeabilization by TDs involves two, apparently. . .

L5 ANSWER 8 OF 18 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:143575 BIOSIS
DOCUMENT NUMBER: PREV199800143575
TITLE: Mercuric compounds inhibit human monocyte function by inducing apoptosis: Evidence for formation of reactive oxygen species, development of mitochondrial membrane permeability transition and loss of reductive reserve.
AUTHOR(S): Insug, O.; Datar, Sugandha; Koch, Cameron J.; Shapiro, Irving M.; Shenker, Bruce J. (1)
CORPORATE SOURCE: (1) Dep. Pathol., Univ. Pa., Sch. Dental Med., 4010 Locust St., Philadelphia, PA 19104-6002 USA
SOURCE: Toxicology, (Dec. 31, 1997) Vol. 124, No. 3, pp. 211-224.
ISSN: 0300-483X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The focus of this investigation was to examine the effects of low concentrations of organic mercuric **compounds** on human monocyte function and to relate these effects to apoptosis. Following exposure of monocytes to 0-5 muM MeHgCl, phagocytic. . . monocyte death was due to apoptosis, a number of flow cytometric studies were performed. Mercury-treated cells exhibited increased Hoechst 33258

fluorescence, while maintaining their ability to exclude the vital **dye** 7-aminoa[redacted]nomycin D. Furthermore, monocy[redacted] exhibited changes in light scatter patterns that were consistent with apoptosis; these included decreased forward light. . . . mercury. Mercury-treated cells also exhibited changes in lipid organization within the plasma membrane as evidenced by increased uptake of the **fluorescent** probe, merocyanine 540, and by elevated annexin V binding to phosphatidylserine. Using the **fluorescent** probes DiOC6(3) and rhodamine 123 we noted that within 1 h of exposure to mercury, monocytes exhibited a decrease in mitochondrial **transmembrane potential** (PSIm). Since a decreased PSIm is associated with altered mitochondrial function, the hypothesis that mercury potentiated reactive oxygen species (ROS). . . . was tested. We noted that treated cells generated ROS, as evidenced by oxidation of hydroethidine and the generation of the **fluorescent** product, ethidium. Finally, since ROS would also lower monocyte reductive reserve, we also measured GSH levels in mercury-treated cells. Chemical. . .

L5 ANSWER 9 OF 18 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1984:206370 BIOSIS
DOCUMENT NUMBER: BA77:39354
TITLE: CORRELATION OF INCREASED INTRA ACROSOMAL PH WITH THE HAMSTER SPERM ACROSOME REACTION.
AUTHOR(S): WORKING P K; MEIZEL S
CORPORATE SOURCE: CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY, P. O. BOX 12137, RESEARCH TRIANGLE PARK, N.C. 27709.
SOURCE: J EXP ZOOL, (1983) 227 (1), 97-108.
CODEN: JEZOAO. ISSN: 0022-104X.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB. . . mM) at 4 h did not stimulate AR over control levels, suggesting that the stimulation of AR by the other **compounds** was not directly due to depletion of acrosomal ATP or alteration of the acrosomal **transmembrane potential**. The AR also was not stimulated by either DCCD or FCCP added prior to 3 h of incubation of sperm, whereas both **compounds** were increasingly effective at stimulating AR with increasing length of preincubation of sperm before the addition of the test **compounds**. The intraacrosomal pH of sperm incubated in low [K+] (0.6-0.9 mM) for 3.5 h rose by least 1 pH unit (as measured with the **fluorescent dye** 9-aminoacridine) within 15-30 min after raising extracellular [K+] to 4.2-4.5 mM. The pH rise occurred even in the presence of. . .

L5 ANSWER 10 OF 18 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002112973 EMBASE
TITLE: Mitochondrial and nonmitochondrial reduction of MTT: Interaction of MTT with TMRE, JC-1, and NAO mitochondrial fluorescent probes.
AUTHOR: Bernas T.; Dobrucki J.
CORPORATE SOURCE: J. Dobrucki, Department of Biophysics, Institute of Molecular Biology, Jagiellonian University, ul. Gronostajowa 7, 30-387 Krakow, Poland. dobrucki@mol.uj.edu.pl
SOURCE: Communications in Clinical Cytometry, (1 Apr 2002) 47/4 (236-242).
Refs: 35
ISSN: 0196-4763 CODEN: CCCYEM
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . MTT, we imaged the formation of MTT-formazan deposits using

backscattered light confocal microscopy. Mitochondria were visualized in viable cells using **fluorescent dyes** that bind in a manner dependent (JC-1 and TMRE) or independent (NAO) of mitochondrial electric **potential**. Results: Only 25-45% of MTT-formazan was associated with mitochondria after 25 min of incubation. No more than 25% of the mitochondrial area on images was occupied by MTT-formazan. Mitochondrial **fluorescence** of TMRE, NAO, and the monomeric form of JC-1 decreased rapidly in cells incubated with MTT. However, the intensity of **fluorescence** of JC-1 aggregates dropped by less than 30% at the onset of incubation and remained constant as reduction of MTT. . . . well as TMRE and NAO, accumulating in mitochondria may be displaced by MTT. Thus, the presence of positively charged organic **compounds** (like MTT) may distort measurements of mitochondrial **transmembrane** electric **potential**, which are based on accumulation of **fluorescent dyes**. .COPYRGT. 2002 Wiley-Liss, Inc.

L5 ANSWER 11 OF 18 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001405434 EMBASE
TITLE: Mitochondrial injury by disulfiram: Two different mechanisms of the mitochondrial permeability transition.
AUTHOR: Balakirev M.Y.; Zimmer G.
CORPORATE SOURCE: M.Y. Balakirev, Institut de Biologie Structurale, 41 rue Jules Horowitz, 38027 Grenoble, France. maxbala@ibs.fr
SOURCE: Chemico-Biological Interactions, (21 Dec 2001) 138/3 (299-311).
Refs: 47
ISSN: 0009-2797 CODEN: CBINA8
PUBLISHER IDENT.: S 0009-2797(01)00283-6
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Disulfiram (Ds), a clinically employed alcohol deterrent of the thiuram disulfide (TD) class of **compounds**, is known to cause hepatitis and neuropathies. Although this drug has been shown to inhibit different thiol-containing enzymes, the actual. . . GSH-dependent manner. At the concentration above characteristic threshold, TDs induced irreversible oxidation of NAD(P)H and glutathione (GSH) pools, collapse of **transmembrane potential**, and inhibition of oxidative phosphorylation. The presence of Ca(2+) and exhaustion of mitochondrial glutathione (GSH + GSSG) decreased the threshold concentration of TDs. Swelling of the mitochondria and leakage of non-transported **fluorescent dye** BCECF from the matrix indicated that TDs induced the mitochondrial permeability transition (MPT). Mitochondrial permeabilization by TDs involves two, apparently. . .

L5 ANSWER 12 OF 18 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999088314 EMBASE
TITLE: Poly(ADP-ribose) synthetase activation mediates mitochondrial injury during oxidant-induced cell death.
AUTHOR: Virag L.; Salzman A.L.; Szabo C.
CORPORATE SOURCE: Dr. C. Szabo, Division of Critical Care, Children's Hospital Medical Center, 3333 Burnet Ave., Cincinnati, OH 45229, United States. szabocsaba@aol.com
SOURCE: Journal of Immunology, (1 Oct 1998) 161/7 (3753-3759).
Refs: 52
ISSN: 0022-1767 CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . investigated whether PARS activation contributes to the

mitochondrial alterations in cells exposed to oxidants. Authentic peroxynitrit[redacted]20 .mu.M), the peroxynitrite-g[redacted]rating **compound** 3-morpholinosid-nonimine, the combination of pyrogallol and S-nitroso-N-acetyl-D,L-penicillamine, as well as hydrogen peroxide induced a time-and dose-dependent decrease in mitochondrial **transmembrane potential** (.DELTA..OMEGA.(m)) in thymocytes, as determined by flow cytometry using the mitochondrial **potential** sensitive **dyes** DiOC6(3) and JC-1. A time- and dose-dependent increase in secondary reactive oxygen intermediate production and loss of cardiolipin, an indicator of mitochondrial membrane damage, were also observed, as measured by flow cytometry using the **fluorescent dyes** dihydroethidine and nonyl-acridine orange, respectively. Inhibition of PARS by 3-aminobenzamide or 5-iodo-6- amino-1,2-benzopyrone attenuated peroxynitrite-induced .DELTA..OMEGA.(m) reduction, secondary reactive oxygen. . .

L5 ANSWER 13 OF 18 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 1998038619 EMBASE

TITLE: Mercuric compounds inhibit human monocyte function by inducing apoptosis: Evidence for formation of reactive oxygen species, development of mitochondrial membrane permeability transition and loss of reductive reserve.

AUTHOR: InSug O.; Datar S.; Koch C.J.; Shapiro I.M.; Shenker B.J.

CORPORATE SOURCE: B.J. Shenker, Department of Pathology, University of Pennsylvania, School of Dental Medicine, 4010 Locust Street, Philadelphia, PA 19104-6002, United States. shenker@path.dental.upenn.edu

SOURCE: Toxicology, (31 Dec 1997) 124/3 (211-224).
Refs: 39
ISSN: 0300-483X CODEN: TXCYAC

PUBLISHER IDENT.: S 0300-483X(97)00153-4

COUNTRY: Ireland

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 026 Immunology, Serology and Transplantation
052 Toxicology

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The focus of this investigation was to examine the effects of low concentrations of organic mercuric **compounds** on human monocyte function and to relate these effects to apoptosis. Following exposure of monocytes to 0-5 .mu.M MeHgCl, phagocytic. . . monocyte death was due to apoptosis, a number of flow cytometric studies were performed. Mercury-treated cells exhibited increased Hoechst 33258 **fluorescence**, while maintaining their ability to exclude the vital **dye** 7-aminoactinomycin D. Furthermore, monocytes exhibited changes in light scatter patterns that were consistent with apoptosis; these included decreased forward light. . . mercury. Mercury-treated cells also exhibited changes in lipid organization within the plasma membrane as evidenced by increased uptake of the **fluorescent** probe, merocyanine 540, and by elevated annexin V binding to phosphatidylserine. Using the **fluorescent** probes DiOC6(3) and rhodamine 123 we noted that within 1 h of exposure to mercury, monocytes exhibited a decrease in mitochondrial **transmembrane potential** (.PSI.(m)). Since a decreased .PSI.(m) is associated with altered mitochondrial function, the hypothesis that mercury potentiated reactive oxygen species (ROS). . . was tested. We noted that treated cells generated ROS, as evidenced by oxidation of hydroethidine and the generation of the **fluorescent** product, ethidium. Finally, since ROS would also lower monocyte reductive reserve, we also measured GSH levels in mercury-treated cells. Chemical. . .

L5 ANSWER 14 OF 18 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:310773 CAPLUS
TITLE: Mitochondrial and nonmitochondrial reduction of MTT: interaction of MTT with TMRE, JC-1, and NAO mitochondrial fluorescent probes
AUTHOR(S): Bernas, Tytus; Dobrucki, Jurek
CORPORATE SOURCE: Laboratory of Confocal Microscopy and Image Analysis, Jagiellonian University, Krakow, 30-387, Pol.
SOURCE: Cytometry (2002), 47(4), 236-242
CODEN: CYTODQ; ISSN: 0196-4763
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

AB Background: Bioredn. of water-sol. tetrazolium salts (e.g., MTS, XTT, and MTT) to their resp. formazans is generally regarded as an indicator of cell "redox activity.". The reaction is attributed mainly to mitochondrial enzymes and electron carriers. However, MTT redn. may also be catalyzed by a no. of other nonmitochondrial enzymes. The goal of this work was to establish the sites of MTT redn. in intact HepG2 human hepatoma cells in culture. Methods: In order to establish the subcellular localization of the sites of redn. of MTT, we imaged the formation of MTT-formazan deposits using backscattered light confocal microscopy. Mitochondria were visualized in viable cells using **fluorescent dyes** that bind in a manner dependent (JC-1 and TMRE) or independent (NAO) of mitochondrial elec. **potential**. Results: Only 25-45% of MTT-formazan was assocd. with mitochondria after 25 min of incubation. No more than 25% of the mitochondrial area on images was occupied by MTT-formazan. Mitochondrial **fluorescence** of TMRE, NAO, and the monomeric form of JC-1 decreased rapidly in cells incubated with MTT. However, the intensity of **fluorescence** of JC-1 aggregates dropped by less than 30% at the onset of incubation and remained const. as redn. of MTT proceeded further. Conclusions: (1) Most of MTT-formazan deposits are not coincident with mitochondria. (2) Monomeric JC-1, as well as TMRE and NAO, accumulating in mitochondria may be displaced by MTT. Thus, the presence of pos. charged org. **compds**. (like MTT) may distort measurements of mitochondrial **transmembrane** elec. **potential**, which are based on accumulation of **fluorescent dyes**.

L5 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:856684 CAPLUS
DOCUMENT NUMBER: 136:194025
TITLE: Mitochondrial injury by disulfiram: two different mechanisms of the mitochondrial permeability transition
AUTHOR(S): Balakirev, Maxim Yu; Zimmer, Guido
CORPORATE SOURCE: Institut de Biologie Structurale, Grenoble, 38027, Fr.
SOURCE: Chemico-Biological Interactions (2001), 138(3), 299-311
CODEN: CBINA8; ISSN: 0009-2797
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

AB Disulfiram (Ds), a clin. employed alc. deterrent of the thiuram disulfide (TD) class of **compds**., is known to cause hepatitis and neuropathies. Although this drug has been shown to inhibit different

thiol-contg. enzymes, the actual mechanism of Ds toxicity is not clear. The authors have previously demonstrated that Ds impairs the permeability of inner mitochondrial membrane (1998). In this report, the effect of Ds and its structural analog thiram (Th) on mitochondrial functions was studied in detail. The authors found that mitochondria metabolize TDs in a NAD(P)H- and GSH-dependent manner. At the concn. above characteristic threshold, TDs induced irreversible oxidn. of NAD(P)H and glutathione (GSH) pools, the collapse of **transmembrane potential**, and inhibition of oxidative phosphorylation. The presence of Ca2+ and exhaustion of mitochondrial glutathione (GSH+GSSG) decreased the threshold concn. of TDs. Swelling of the mitochondria and leakage of non-transported **fluorescent dye** BCECF from the matrix indicated that TDs induced the mitochondrial permeability transition (MPT). Mitochondrial permeabilization by TDs involves 2, apparently distinct mechanisms. In the presence of Ca2+, TDs produced cylosporin A-sensitive swelling of mitochondria, which was inhibited by ADP and accelerated by carboxyatractyloside (CATR) and phosphate. In contrast, the swelling produced by TDs in the absence of Ca2+ was not sensitive to cyclosporin A (CsA), ADP, and CATR but was inhibited by phosphate. Titrn. with N-ethylmaleimide revealed that these 2 mechanisms involve different SH-groups and probably different transport proteins on the IMM. The findings indicate that at pharmacol. relevant concns. TDs may cause an irreversible mitochondrial injury as a result of induction of the MPT.

L5 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:652194 CAPLUS
DOCUMENT NUMBER: 130:24077
TITLE: Poly(ADP-ribose) synthetase activation mediates mitochondrial injury during oxidant-induced cell death
AUTHOR(S): Virag, Laszlo; Salzman, Andrew L.; Szabo, Csaba
CORPORATE SOURCE: Division of Critical Care, Children's Hospital Medical Center, Cincinnati, OH, 45229, USA
SOURCE: Journal of Immunology (1998), 161(7), 3753-3759
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Reactive oxidant species are important mediators of tissue injury in shock, inflammation, and reperfusion injury. The actions of a no. of these oxidants (e.g., hydroxyl radical and peroxynitrite, a reactive oxidant produced by the reaction of nitric oxide and superoxide) are mediated in part by the activation of the nuclear nick sensor enzyme, poly(ADP)-ribose synthetase (PARS), with consequent cellular energy depletion. Here the authors investigated whether PARS activation contributes to the mitochondrial alterations in cells exposed to oxidants. Authentic peroxynitrite (20 .mu.M), the peroxynitrite-generating **compd**. 3-morpholinosidnonimine, the combination of pyrogallol and S-nitroso-N-acetyl-D,L-penicillamine, as well as hydrogen peroxide induced a time- and dose-dependent decrease in mitochondrial **transmembrane potential** (.DELTA..PSI.m) in thymocytes, as detd. by flow cytometry using the mitochondrial **potential** sensitive **dyes** DiOC6(3) and JC-1. A time- and dose-dependent increase in secondary reactive oxygen intermediate prodn. and loss of cardiolipin, an indicator of mitochondrial membrane damage, were also obsd., as measured by flow cytometry using the **fluorescent dyes** dihydroethidine and nonyl-acridine orange, resp. Inhibition of PARS by

3-aminobenzamide or 5-iodo-6-amino-1,2-benzopyrone attenuated peroxynitrite-induced .DELTA..PSI.m redn., secondary reactive oxygen intermediate generation, cardiolipin degrdn., and intracellular calcium mobilization. Furthermore, thymocytes from PARS-deficient animals were protected against the peroxynitrite- and hydrogen peroxide-induced functional and ultrastructural mitochondrial alterations. Thus, mitochondrial perturbations during oxidant-mediated cytotoxicity are related to PARS activation rather than to direct effects of the oxidants on the mitochondria.

L5 ANSWER 17 OF 18 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:72844 CAPLUS
DOCUMENT NUMBER: 128:177031
TITLE: Mercuric compounds inhibit human monocyte function by inducing apoptosis: evidence for formation of reactive oxygen species, development of mitochondrial membrane permeability transition and loss of reductive reserve
AUTHOR(S): InSug, O.; Datar, Sugandha; Koch, Cameron J.; Shapiro, Irving M.; Shenker, Bruce J.
CORPORATE SOURCE: University of Pennsylvania, Department of Pathology, School of Dental Medicine and School of Medicine, 4010 Locust Street, Philadelphia, PA, 19104-6002, USA
SOURCE: Toxicology (1997), 124(3), 211-224
CODEN: TXCYAC; ISSN: 0300-483X
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The focus of this investigation was to examine the effects of low concns. of org. mercuric **compds**. on human monocyte function and to relate these effects to apoptosis. Following exposure of monocytes to 0-5 .mu.M MeHgCl, phagocytic function and capacity to generate a respiratory burst, following PMA activation, were detd. The authors found that the mercury-treated cells exhibited reduced phagocytic activity. Exposure to the same mercury concn. range, also caused a marked increase in cell death. To ascertain if monocyte death was due to apoptosis, a no. of flow cytometric studies were performed. Mercury-treated cells exhibited increased Hoechst 33258 **fluorescence**, while maintaining their ability to exclude the vital **dye** 7-aminoactinomycin D. Furthermore, monocytes exhibited changes in light scatter patterns that were consistent with apoptosis; these included decreased forward light scatter and increased side scatter. The percentage of cells undergoing apoptosis was dependent upon the mercury content of the medium, regardless of whether the metal was present as Me, Et or Ph mercury. Mercury-treated cells also exhibited changes in lipid organization within the plasma membrane as evidenced by increased uptake of the **fluorescent** probe, merocyanine 540, and by elevated annexin V binding to phosphatidylserine. Using the **fluorescent** probes DiOC6(3) and rhodamine 123 the authors noted that within 1 h of exposure to mercury, monocytes exhibited a decrease in mitochondrial **transmembrane potential**. Since a decreased mitochondrial **transmembrane potential** is assocd. with altered mitochondrial function, the hypothesis that mercury potentiated reactive oxygen species (ROS) generation and that these species promoted apoptosis was tested. The authors noted that treated cells generated ROS, as evidenced by oxidn. of hydroethidine and the generation of the **fluorescent** product, ethidium. Finally, since ROS would also lower monocyte reductive reserve, we also measured GSH levels in mercury-treated cells. Chem. measurement of GSH indicated that there was thiol depletion. The authors suggest that

the low thiol reserve predisposes cells to ROS damage and at the same time
activates death-signaling pathways.

L5 ANSWER 18 OF 18 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:502904 CAPLUS
DOCUMENT NUMBER: 99:102904
TITLE: Correlation of increased intraacrosomal pH with the hamster sperm acrosome reaction
AUTHOR(S): Working, Peter K.; Meizel, Stanley
CORPORATE SOURCE: Sch. Med., Univ. California, Davis, CA, 95616, USA
SOURCE: J. Exp. Zool. (1983), 227(1), 97-107
CODEN: JEZOAO; ISSN: 0022-104X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Washed cauda epididymal sperm from hamsters were capacitated in vitro in a medium contg. 2 mM Ca2+, 144 mM Na+, and 3 mM K+. Such sperm underwent a significant increase in the no. of acrosomal reactions (AR) within 10 min after the addn. of the Mg2+-ATPase inhibitors DCCD (20 .mu.M) or 4-chloro-7-nitrobenzofuran (10 .mu.M) or the proton ionophore FCCP (6 .mu.g/mL) at 3.5 h of incubation or after addn. of NH4Cl (3 mM) at 4 h of incubation. Addn. of the mitochondrial electron transport inhibitor rotenone (2.5 .mu.M) at 3.5 h or of NaCl (3 mM) or KCl (3 mM) at 4 h did not stimulate AR over control levels, suggesting that the stimulation of AR by the other **compds**. was not directly due to depletion of acrosomal ATP or alteration of the acrosomal **transmembrane potential**. The AR also was not stimulated by either DCCD or FCCP added prior to 3 h of incubation of sperm, whereas both **compds**. were increasingly effective at stimulating AR with increasing length of preincubation of sperm before the addn. of the test **compds**. The intraacrosomal pH of sperm incubation in low K+ concn. (0.6-0.9 mM) for 3.5 h rose by .gtoreq.1 pH unit (as measured with the **fluorescent dye** 9-aminoacridine) within 15-30 min after raising extracellular K+ concn. to 4.2-4.5 mM. The pH rise occurred even in the presence of EGTA (2 mM). Either FCCP (8.mu.g/mL) or DCCD (20 .mu.M), but not rotenone (2.5 .mu.M), plus K+ (3.6 mM), raised the intraacrosomal pH of sperm incubated for 3 h in a low K+ concn. within 10 min after addn. No pH rise occurred in the absence of addnl. K+. Thus, the intraacrosomal pH of the hamster sperm becomes more alk. in a process not requiring high concns. of external Ca2+, but requiring K+. The results of this and previous studies lead to the suggestion that the intraacrosomal pH rise may be mediated via a change in K+ and K+ permeability of sperm head membranes, which allows K+ influx and H+ efflux, and via inhibition of an acrosomal Mg2+-ATPase H+ pump. The permeability changes and the consequent alkalinization of the acrosomal interior may be important steps in late capacitation and/or the mammalian AR.

=> d l6 ibib kwic

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:90336 CAPLUS
DOCUMENT NUMBER: 136:147469
TITLE: Ion channel assay methods using electrical stimulation
INVENTOR(S): Maher, Michael P.; Gonzalez, Jesus E., III
PATENT ASSIGNEE(S): Aurora Biosciences Corporation, USA
SOURCE: PCT Int. Appl., 146 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002008748	A2	20020131	WO 2001-US21652	20010709
WO 2002008748	A3	20020502		

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002025568	A1	20020228	US 2001-804480	20010312
US 2002028480	A1	20020307	US 2001-804580	20010312
US 2002045159	A1	20020418	US 2001-804457	20010312

PRIORITY APPLN. INFO.:	US 2000-217219P	P	20000710
	US 2000-217221P	P	20000710
	US 2000-217666P	P	20000710
	US 2000-217671P	P	20000710
	US 2001-804457	A	20010312
	US 2001-804458	A	20010312
	US 2001-804480	A	20010312
	US 2001-804580	A	20010312

AB A method of characterizing the biol. activity of a candidate **compd**. may include exposing cells to the candidate **compd**., and then exposing the cells to a repetitive application of elec. fields so as to set the **transmembrane potential** to a level corresponding to a pre-selected voltage dependent state of a target ion channel. Adherent RBL cells, endogenously expressing the potassium inward rectifier channel IRK1, were seeded into 96-well plates and loaded with **FRET** dyes. Three rows of wells contained 400 .mu.M barium chloride to block the IRK1 channel. The plates were analyzed using a VIPR reader while being elec. stimulated with a biphasic stimulus train repeated at a frequency of 50 Hz and with a 5 ms/phase pulse duration.

=> d his

(FILE 'HOME' ENTERED AT 08:37:33 ON 07 OCT 2002)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 08:38:04 ON 07 OCT 2002

L1 270 S MAHER M/AU
L2 2354 S GONZALEZ J/AU
L3 0 S L1 AND L2
L4 0 S TRANSMEMBRANE (P) POTENTIAL (P) OMPOUND (P) FLUORES? (P) DYE
L5 18 S TRANSMEMBRANE (P) POTENTIAL (P) COMPOUND (P) FLUORES? (P) DYE
L6 1 S TRANSMEMBRANE (P) POTENTIAL (P) COMPOUND (P) FRET

=> s transmembrane (p) potential (p) screen? (p) FRET

L7 1 TRANSMEMBRANE (P) POTENTIAL (P) SCREEN? (P) FRET

=> d l7 ibib kwic

L7 ANSWER 1 OF [redacted] CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:435043 CAPLUS
DOCUMENT NUMBER: 135:43136
TITLE: Detection of transmembrane potentials by fluorescent resonance energy transfer (FRET) between a hydrophobic fluorescent ion and a chromophore
INVENTOR(S): Tsien, Roger Y.; Gonzalez, Jesus E. III
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 154 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001042211	A2	20010614	WO 2000-US33739	20001212
WO 2001042211	A3	20020117		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-459956 A 19991213
OTHER SOURCE(S): MARPAT 135:43136
IT Animal tissue culture
Cell membrane
Chromophores
Cyanine dyes
Drug **screening**
Endoplasmic reticulum
Hydrophobicity
Solubilizers
Test kits
(detection of **transmembrane potentials** by fluorescent resonance energy transfer (**FRET**) between a hydrophobic fluorescent ion and a chromophore)

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	72.27	72.48

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-3.72	-3.72

STN INTERNATIONAL LOGOFF AT 08:43:36 ON 07 OCT 2002

L Number	Hits	Search Text	DB	Time stamp
1	2	transmembrane same potential same compound same FRET	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/07 08:30
2	2	wo adj "9641166"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/07 08:30
3	19	transmembrane same potential same compound same fluorescent same dye	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/07 08:33
4	3	Maher-m-p.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/07 08:34
5	4	gonzalez-j-e.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/07 08:34